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NEWS	22	APR 28	IMSRSEARCH reloaded with enhancements
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NEWS	25	JUN 06	EPFULL enhanced with 260,000 English abstracts
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NEWS	29	JUN 25	CA/CAPLUS and USPAT databases updated with IPC reclassification data
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=> S (human or sapien) (3A) (kallikrein)

L1 5997 (HUMAN OR SAPIEN) (3A) (KALLIKREIN)

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L2 564 L1 (8A) (INHIBITOR OR INHIBIT OR INHIBITING OR INHIBITED OR INHIBITION OR MODULATOR OR MODULATE OR MODULATING OR MODULATED OR MODULATION)

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L3 445 L1 (6A) (METHOD OR ANALYSIS OR ANALYZE OR ANALYZED OR ANALYZING OR TEST OR TESTING OR TESTED OR DETERMINING OR DETERMINATION OR DETERMINE OR DETERMINED OR EVALUATE OR EVALUATION OR EVALUATED OR EVALUATION)

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L5 ANSWER 1 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN  
AN 2005:823838 CAPLUS  
DN 143:222436  
TI Use of human kallikrein 2 (KLK2) polypeptides and polynucleotides in drug screening and in diagnosis of various disorders  
IN Goltz, Stefan; Brueggemeier, Ulf; Geerts, Andreas; Summer, Holger  
PA Bayer Healthcare A.-G., Germany  
SO PCT Int. Appl., 96 pp.  
CODEN: PIXXD2  
DT Patent  
LA English  
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2005075662	A2	20050818	WO 2005-EP342	20050115
	WO 2005075662	A3	20051103		
	W:	AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW			
	RW:	BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IS, IT, LT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG			
	EP 1711828	A2	20061018	EP 2005-700936	20050115
	R:	AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, FI, RO, CY, TR, BG, CZ, EE, HU, PL, SK, IS			
	US 20070218047	A1	20070920	US 2007-587452	20070119
PRAI	EP 2004-1737	A	20040128		
	WO 2005-EP342	W	20050115		

AB The invention provides a method of screening for compds. useful in the treatment of hematomol. disorders, cardiovascular diseases, cancer, inflammation, neurol. diseases, reproduction disorders and urol. diseases, which involves the use of human kallikrein 2 (KLK2) polypeptides and polynucleotides. Specifically, the drug screening assays look for the ability of a compound to bind said KLK2 polypeptides and polynucleotides, and modulate the activity of KLK2. The invention also provides a pharmaceutical composition comprising said KLK2 modulator and its use in treatment of disclosed diseases and/or disorders. The invention relates said KLK2 modulator may a small mol., a RNA mol., an antisense oligonucleotide, a polypeptide, an antibody or a ribozyme. The invention further provides a method for preparing said pharmaceutical composition

Finally,  
the invention provides a method for diagnosing said diseases and/or disorder which involves determining the presence and/or expression of said KLK2

polynucleotides. In the examples, the invention presented the relative expression of KLK2 mRNA in various human tissues and cells, with the greatest expression being detected in the prostate.

L5 ANSWER 2 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN  
 AN 2005:528971 CAPLUS  
 DN 143:362636  
 TI An Effective Method for Isolating Human Urinary  
 Kallikrein and Trypsin Inhibitor  
 AU Wang, Ju; Hong, An; Pu, Hanlin; Chen, Xiaojia  
 CS Bio-engineering Institute, Jinan University, Guangzhou, Guangdong  
 Province, 510632, Peop. Rep. China  
 SO Zhongguo Yiyao Gongye Zazhi (2004), 35(7), 401-403  
 CODEN: ZYGZEA; ISSN: 1001-8255  
 PB Zhongguo Yiyao Gongye Zazhi Bianjibu  
 DT Journal  
 LA Chinese  
 AB The human urinary kallikrein (hKN) and human  
 urinary trypsin inhibitor (hUTI) are two acidic proteins in the  
 urine. The method of ethanol precipitation together with ZnCl2 colloid  
 precipitation was  
 found to isolate them with higher yield and activities.

L5 ANSWER 3 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN  
 AN 2003:239423 BIOSIS  
 DN PREV200300239423  
 TI DX-88 a recombinant inhibitor of human plasma  
 kallikrein. Evaluation of efficacy and safety in  
 patients with hereditary angioedema.  
 AU Williams, T.; Cicardi, M. [Reprint Author]; Bork, K.; Gonzalez-Quevedo,  
 T.; Caballero, T.  
 CS Dipartimento di Medicina Interna, Universita di Milano, IRCCS Ospedale  
 Maggiore, Milano, Italy  
 SO Journal of Allergy and Clinical Immunology, (April 2003) Vol. 111, No. 4,  
 pp. 908. print.  
 Meeting Info.: 60th Anniversary Meeting of the American Academy of  
 Allergy, Asthma and Immunology (AAAAI). Denver, CO, USA. March 07-12,  
 2003. American Academy of Allergy, Asthma and Immunology.  
 CODEN: JACIBY. ISSN: 0091-6749.  
 DT Conference; (Meeting)  
 Conference; Abstract; (Meeting Abstract)  
 LA English  
 ED Entered STN: 21 May 2003  
 Last Updated on STN: 21 May 2003

L5 ANSWER 4 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN  
 AN 2002:830198 CAPLUS  
 DN 137:291281  
 TI Complex of human kallikrein 2 (hK2) and protease  
 inhibitor-6 (PI-6) in prostate tumor tissue and methods of using  
 the complex and its constituents  
 IN Mikolajczyk, Stephen D.; Saedi, Mohammad S.  
 PA Hyrbritech Incorporated, USA  
 SO U.S., 19 pp., Cont.-in-part of U.S. 6,284,873.  
 CODEN: USXXAM  
 DT Patent  
 LA English  
 FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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PI	US 6472143	B1	20021029	US 2000-667288	20000922

US 6284873	B1	20010904	US 1999-266957	19990312
US 20010031860	A1	20011018	US 2001-882648	20010615
PRAI US 1999-266957	A2	19990312		

AB The present invention provides a novel complex of hK2 and PI-6 and methods of using the novel complex and its constituents. The novel complexes of hK2 and PI-6 of the present invention and the PI-6 exist at an elevated level in prostate cancer tissues. PI-6 also exists at an elevated level in other types of cancer cells. Therefore, the hK2-PI6 complexes and PI-6 of the present invention may be used as a serum marker for detecting cancers, particularly prostate cancer. They may also be used as an immunohistol. marker to detect prostate cancer tissues. In accordance with the present invention, the hK2-PI6 complexes of the present invention may be detected in patient tissue samples by immunohistochem. and/or in patient fluid samples by in vitro immunoassay procedures. Diagnostic methods for detecting the existence of prostate cancer is also provided.

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 5 OF 22 CAPLUS COPYRIGHT 2008 ACS ON STN  
AN 2000:666861 CAPLUS  
DN 133:250893

TI A novel complex of human kallikrein 2 (hK2) and  
protease inhibitor-6 (PI-6) in prostate tumor tissue and methods  
of using the complex for detecting prostate cancer

IN Mikolajczyk, Stephen; Saedi, Mohammad

PA Hybritech Incorporated, USA

SO PCT Int. Appl., 49 pp.

CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000055308	A1	20000921	WO 2000-US1937	20000126
	W: AU, CA, JP				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	US 6284873	B1	20010904	US 1999-266957	19990312
	CA 2365540	A1	20000921	CA 2000-2365540	20000126
	EP 1159411	A1	20011205	EP 2000-905737	20000126
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
	JP 2002538818	T	20021119	JP 2000-605726	20000126
	AU 778038	B2	20041111	AU 2000-27376	20000126
	US 20010031860	A1	20011018	US 2001-882648	20010615
PRAI	US 1999-266957	A	19990312		
	WO 2000-US1937	W	20000126		

AB The present invention provides a novel complex of hK2 and PI-6 and methods of using the novel complex. The novel complexes of hK2 and PI-6 of the present invention exist at an elevated level in prostate cancer tissues. Therefore, the hK2-PI6 complexes of the present invention may be used as a serum marker for detecting prostate cancer. They may also be used as an immunohistol. marker to detect prostate cancer tissues. In accordance with the present invention, the hK2-PI6 complexes of the present invention may be detected in patient tissue samples by immunohistochem. and/or in patient fluid samples by in vitro immunoassay procedures. Diagnostic kits and diagnostic methods for detecting the existence of prostate cancer are also provided.

RE.CNT 6 THERE ARE 6 CITED REFERENCES AVAILABLE FOR THIS RECORD  
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 6 OF 22 MEDLINE on STN DUPLICATE 1  
 AN 1999384018 MEDLINE  
 DN PubMed ID: 10454435  
 TI Kallikrein gene delivery inhibits vascular smooth muscle cell growth and neointima formation in the rat artery after balloon angioplasty.  
 AU Murakami H; Yayama K; Miao R Q; Wang C; Chao L; Chao J  
 CS Department of Biochemistry and Molecular Biology, Medical University of South Carolina, Charleston, SC, USA.  
 NC HL-29397 (United States NHLBI)  
 HL-52196 (United States NHLBI)  
 SO Hypertension, (1999 Aug) Vol. 34, No. 2, pp. 164-70.  
 Journal code: 7906255. ISSN: 0194-911X.  
 CY United States  
 DT (COMPARATIVE STUDY)  
 (IN VITRO)  
 Journal; Article; (JOURNAL ARTICLE)  
 (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)  
 LA English  
 FS Priority Journals  
 EM 199909  
 ED Entered STN: 25 Sep 1999  
 Last Updated on STN: 3 Mar 2000  
 Entered Medline: 15 Sep 1999  
 AB Tissue kallikrein cleaves kininogen substrate to produce vasoactive kinin peptides that have been implicated in the proliferation of vascular smooth muscle cells (VSMCs). To explore potential roles of the kallikrein-kinin system in vascular biology, we evaluated the effects of adenovirus-mediated human kallikrein gene delivery on the growth of primary cultured VSMCs and in balloon-injured rat artery in vivo. Kallikrein gene transfer into cultured rat VSMCs resulted in time-dependent secretion of recombinant human tissue kallikrein and inhibition of cell proliferation. Balloon angioplasty reduced endogenous rat tissue kallikrein mRNA and protein levels at the injured site. In rats that received adenovirus-mediated human kallikrein gene delivery, we observed a 39% reduction in intima/media ratio at the injured vessel after delivery compared with that of rats that received control virus (n=8, P<0.01). Icatibant, a specific bradykinin B(2) receptor antagonist, blocked the protective effect and reversed the intima/media ratio to that of the control rats (n=5, P<0.01). After gene delivery, human kallikrein mRNA was identified at the injured vessel and a 3-fold increase occurred in kininogenase activity. cAMP and cGMP levels in balloon-injured aorta increased significantly at 4, 7, and 14 days after kallikrein gene delivery, but icatibant abolished the increase. These results provide new insights into the role of the vascular kallikrein-kinin system and have significant implications for gene therapy to treat restenosis or atherosclerosis.

L5 ANSWER 7 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN  
 AN 1999:717391 CAPLUS  
 DN 132:104485  
 TI Purification of human urinary kallikrein from residue of urinary trypsin inhibitor production  
 AU He, Guozhen; Tang, Liyun; Zheng, Lijun  
 CS Guangdong Yantang Biochemistry Pharmaceutical Co., Ltd., Canton, 510507, Peop. Rep. China  
 SO Yaowu Shengwu Jishu (1999), 6(2), 103-106  
 CODEN: YSJIFO; ISSN: 1005-8915  
 PE Yaowu Shengwu Jishu Bianjibu  
 DT Journal  
 LA Chinese

AB The purification method of human urinary kallikrein (HUKN) combined with urinary trypsin inhibitor was reported. HUKN had been purified by use of CM-Sepharose FF, DEAE-Sepharose FF and Aprotinin-Sepharose CL-4B chromatog. The purified HUKN with a specific activity of 293.9 EU/mg protein and the recovery rate of 48.6% was a single peak on HPLC. The technol. is simple, and can be scaled up easily.

L5 ANSWER 8 OF 22 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN DUPLICATE 2

AN 1995039278 EMBASE

TI Proteinases from the fibrinolytic and coagulation systems: Analyses of binding to pregnancy zone protein, a pregnancy-associated plasma proteinase inhibitor.

AU Earbelaer, L.; Jensen, P.E.H.; Stigbrand, T. (correspondence)

CS Dept. Medical Biochemistry/Biophys., University of Umea, S-901 87 Umea, Sweden.

SO Fibrinolysis, (1995) Vol. 9, No. 1, pp. 41-47.

ISSN: 0268-9499 CODEN: FBRIE7

CY United Kingdom

DT Journal; Article

FS 025 Hematology

029 Clinical and Experimental Biochemistry

037 Drug Literature Index

LA English

SL English

ED Entered STN: 8 Mar 1995

Last Updated on STN: 8 Mar 1995

AB The inhibitory effects of pregnancy zone protein (PZP) on proteinases within the fibrinolytic and coagulation systems have been studied and compared to that of human  $\alpha(2)$ -macroglobulin ( $\alpha(2)$ -M). Plasmin, t-PA, urokinase, thrombin, human plasma kallikrein, human and porcine tissue kallikrein were tested for binding to PZP and  $\alpha(2)$ -M. PZP was cleaved at the 'bait' region as seen in SDS-PAGE, by both human and porcine tissue kallikrein, but not by any of the other proteinases tested and we therefore suggest that PZP may have a role in inhibition of tissue kallikrein. Plasmin, thrombin, and plasma kallikrein were found to be bound and inhibited by  $\alpha(2)$ -M by cleavage of the 'bait' regions. Minor amounts of cleavage products of  $\alpha(2)$ -M were detected with t-PA and urokinase after prolonged incubation at room temperature. Cleavage of  $\alpha(2)$ -M was detected following incubation with porcine tissue kallikrein, but no cleavage was seen following incubation with human tissue kallikrein. The fast inhibition of plasmin, thrombin, and plasma kallikrein suggest that  $\alpha(2)$ -M may be physiological relevant as an inhibitor for these proteinases. Despite the similarities between  $\alpha(2)$ -M and PZP, significant differences are observed in the inhibition of proteinases. These results suggest distinctive differences in the function of the two human  $\alpha$ -macroglobulins, PZP does only seem to inhibit human tissue kallikrein of the proteinases tested from the fibrinolytic and coagulation systems.

L5 ANSWER 9 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN

AN 1995:196954 CAPLUS

DN 122:127069

OREF 122:23567a,23570a

TI Measurement of urinary kallikrein activity by kininogenase assay

AU Kasatsuki, Tamako

CS Sch. Med., Iwate Med. Univ., Morioka, 020, Japan

SO Iwate Igaku Zasshi (1994), 46(4), 543-52

CODEN: IIZAXX; ISSN: 0021-3284

DT Journal  
LA Japanese  
AB A method for measurement of urinary kallikrein activity in patients was developed with essential hypertension by kininogenase assay, using com. available reagents. Optimum pH of the activity was 8.5, and Michaelis-Menten constant (Km) value for the activity in bovine low-mol.-weight kininogen was 60 µg kinin/mL and these values were similar to those obtained from purified kallikrein, suggesting that the kininogenase activity represents kallikrein activity. The coefficient of variation was 4.7% for intra-assay and 8.4% for inter-assay, thereby indicating a good reproducibility. Addition of NaCl or albumin into urine sample did not disturb the activity, suggesting that this assay system is available for measurements of the activity in urine obtained from patients with nephrotic syndrome or high Na intakes. When urinary kallikrein excretion was measured by this method in 4 patients with primary aldosteronism, it significantly decreased after excision of adrenal adenomas. This method is therefore useful to assess the precise levels of kallikrein excretion.

L5 ANSWER 10 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN

AN 1993:163754 CAPLUS

DN 118:163754

OREF 118:27949a,27952a

TI Immunoassays for the determination of human tissue kallikrein (TK) in different body fluids based on monoclonal antibodies

AU Witzgall, K.; Godec, G.; Shimamoto, K.; Fink, E.

CS Dep. Clin. Chem., Univ. Munich, Munich, D-8000/2, Germany

SO Agents and Actions Supplements (1992), 38(1, Recent Prog. Kinins: Biochem. Mol. Biol. Kallikrein-Kinin Syst.), 153-8  
CODEN: AASUDJ; ISSN: 0379-0363

DT Journal

LA English

AB A monoclonal antibody produced against human tissue kallikrein was used to develop solid-phase immunoassays for the determination of total immunoreactive tissue kallikrein, the complex of tissue kallikrein with  $\alpha_1$ -proteinase inhibitor ( $\alpha_1$ -antitrypsin), and enzymically active tissue kallikrein. The assays permit the specific determination of various

forms of tissue kallikrein in body fluids and should be very useful in studies on the biol. function of tissue kallikrein-kinin systems.

L5 ANSWER 11 OF 22 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights reserved on STN

AN 1990067249 EMBASE

TI Determination of active human urinary kallikrein and human urokinase (M(r) 33,000 and M(r) 54,000 species) concentration in the presence of cognate serine proteinase.

AU Ascenzi, P.; Amiconi, G.; Menegatti, E.; Guarneri, M.

CS C.N.R., Center for Molecular Biology, Department of Biochemical Sciences, University of Rome 'La Sapienza', Piazzale Aldo Moro 5, 00185 Rome, Italy.  
SO Clinical Chemistry and Enzymology Communications, (1990) Vol. 2, No. 2, pp. 79-85.

ISSN: 0892-2187 CODEN: CCECEY

CY United Kingdom

DT Journal; Article

FS 029 Clinical and Experimental Biochemistry

LA English

SL English

ED Entered STN: 13 Dec 1991

Last Updated on STN: 13 Dec 1991



AB A spectrophotometric method for the determination of active human urinary kallikrein concentration, in the presence of human urokinase (M(r) 33,000 and M(r) 54,000 species), as well as of active human urokinase (M(r) 33,000 and M(r) 54,000 species) concentration, in the presence of human urinary kallikrein, is reported. This rate assay method has been developed from the quantitative analysis of kinetics for the hydrolysis of the N- $\alpha$ -carbobenzoyl-L-lysine p-nitrophenyl ester catalyzed by human urinary kallikrein, in the presence of human urokinase (M(r) 33,000 and M(r) 54,000 species) selectively inhibited by the p-carbomethoxyphenyl ester of the  $\epsilon$ -guanidinocaproic acid methanesulphonate, and by human urokinase (M(r) 33,000 and M(r) 54,000 species), in the presence of human urinary kallikrein selectively inhibited by the bovine basic pancreatic trypsin inhibitor (Kunitz-type inhibitor). The active enzyme concentration has been estimated, under conditions where  $[S](0) > K(m)$ , from the dependence of the initial rate for substrate hydrolysis (i.e.,  $V$ ) on the enzyme concentration (i.e.,  $[E](0)$ ) taking into account the proportionally constant  $k(cat)$  (i.e.,  $V = k(cat).ovrhdot.[E](0)$ ). The minimum active human urinary kallikrein and human urokinase (M(r) 33,000 and M(r) 54,000 species) concentration which may be evaluated from the analysis of the experimental data corresponds to  $3.8 \times 10^{-3}$   $\mu$ M and  $1.4 \times 10^{-2}$   $\mu$ M, respectively.

L5 ANSWER 12 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN

AN 1989:168941 CAPLUS

DN 110:168941

OREF 110:27917a,27920a

TI Use of 125I-aprotinin in the assay of glandular kallikreins

AU Ryan, James W.

CS Dep. Med., Univ. Miami, Miami, FL, 33101, USA

SO Methods in Enzymology (1988), 163(Immunochem. Tech., Pt. M), 160-9

CODEN: MENZAU; ISSN: 0076-6879

DT Journal

LA English

AB The use of 125I-labeled aprotinin as a tracer for labeled inhibitor-enzyme immunoassay of kallikrein of human urine (and other tissues) is described. The assay measures only that fraction of enzyme capable of binding the inhibitor with high affinity; enzyme-bound inhibitor is separated from free inhibitor by the addition of excess immobilized antibodies monospecific for the enzyme.

L5 ANSWER 13 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN

AN 1989:130909 CAPLUS

DN 110:130909

OREF 110:21515a,21518a

TI Correlation of two different assays for urinary kallikrein in normotensive and hypertensive subjects

AU Maier, Manfred; Jerabek, Ingrid; Reissert, Guenther; Hoeltzl, Eva; Binder, Bernd R.

CS Dep. Med. Physiol., Univ. Vienna, Vienna, Austria

SO Clinica Chimica Acta (1988), 178(2), 127-39

CODEN: CCATAR; ISSN: 0009-8981

DT Journal

LA English

AB Possible differences in structure-function relationship of urinary kallikrein between normotensive and hypertensive individuals were analyzed using 2 different assay systems which detect 2 distinct entities of the enzyme. A monospecific goat anti-human urinary kallikrein antibody was characterized by inhibition studies with the purified active enzyme and by trypsin activation of

endogenous urinary prokallikrein. Anal. of the data revealed that the antibody is directed against active kallikrein by recognizing an epitope which is different from the catalytic site of the enzyme but which is being exposed together with the active site during trypsin activation of the proenzyme. A direct RIA for urinary kallikrein was developed and correlated with the kinin-generating activity of the enzyme by assessing endogenous active and trypsin-activated kallikrein in the urine of normotensive and hypertensive subjects. Significant pos. correlations were found between the 2 assays for both active and total kallikrein in normotensive and hypertensive subjects, and the slopes of the resp. regression lines were identical. These data do not provide evidence for a defective enzyme, a defective activation of the proenzyme, or the presence of an inhibitor of urinary kallikrein in essential hypertension.

L5 ANSWER 14 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN

AN 1985:74788 CAPLUS

DN 102:74788

OREF 102:11667a,11670a

TI Reagents for immunoassay of  $\alpha$ 1-proteinase inhibitor-kallikrein complex

PA Sugiura Shinyaku Kaihatsu Kenkyusho K. K., Japan; Maruko Pharmaceutical Co., Ltd.

SO Jpn. Kokai Tokkyo Koho, 7 pp.

CODEN: JKXXAF

DT Patent

LA Japanese

FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	JP 59203958	A	19841119	JP 1983-78158	19830506
PRAI	JP 1983-78158		19830506		

AB An enzyme is bound to antihuman  $\alpha$ 1-proteinase inhibitor antibody and antihuman kallikrein antibody is immobilized on an insol. solid carrier to prepare reagents for use in the immunoassay of  $\alpha$ 1-proteinase inhibitor-kallikrein complex in human body fluids by the sandwich method. Thus,  $\alpha$ 1-proteinase inhibitor and kallikrein in Freund's complete adjuvant were injected into rabbits to form the resp. antibodies. Antikallikrein antibody (40 mg) in 0.1M Na phosphate buffer containing 50 mM NaCl was incubated with polystyrene beads (.apprx.6.5-mm diameter) at 30° for 2 h and then with the same buffer containing 0.1% bovine serum albumin at 30° for 2 h, and the reaction product was kept in the same buffer containing 0.01% thiomersal. Sep., 1.5 mg anti- $\alpha$ 1-proteinase inhibitor antibody in 0.1M Na phosphate buffer containing 50 mM NaCl was incubated with 2% m-maleimidobenzoyl N-hydroxysuccinimide ester in dioxane at 30° for 1 h. After separation on a Sephadex G 25 column, the derivatized antibody was treated with 1.5 mg  $\beta$ -galactosidase at 30° for 1 h to form  $\beta$ -galactosidase-labeled antibody. A 20- $\mu$ L serum sample with addition of 10 mM phosphate buffer containing bovine serum albumin, NaN<sub>3</sub>, MgCl<sub>2</sub>, and NaCl was incubated with the polystyrene beads at 37° for 2 h, followed by incubation with  $\beta$ -galactosidase-labeled antibody. The beads in the buffer were treated with 0.3 mM 4-methylumbelliferyl- $\beta$ -galactoside at 30° for 5-20 min, mixed with 0.1M glycine-NaOH buffer (pH 10.3), and the reaction mixture was analyzed at 450 nm with excitation at 360 nm for measurement of galactosidase for the determination of  $\alpha$ 1-proteinase inhibitor-kallikrein complex.

L5 ANSWER 15 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on STN

AN 1984:231910 BIOSIS

DN PREV198477064894; BA77:64894

TI STUDIES OF ACTIVATED COMPLEMENT C-1 INACTIVATOR PLASMA KALLIKREIN COMPLEX  
IN PURIFIED SYSTEMS AND IN PLASMA QUANTIFICATION BY AN ENZYME LINKED  
DIFFERENTIAL ANTIBODY IMMUNO SORBENT ASSAY.

AU LEWIN M F [Reprint author]; KAPLAN A P; HARPEL P C

CS DIV HEMATOLOGY-ONCOLOGY, DEP MED, NY HOSP CORNELL MED CENTER, NEW YORK, NY  
10021, USA

SO Journal of Biological Chemistry, (1983) Vol. 258, No. 10, pp. 6415-6421.  
CODEN: JBCHA3. ISSN: 0021-9258.

DT Article

FS BA

LA ENGLISH

AB An enzyme-linked immunosorbent assay (ELISA) was developed for the  
quantification of C.hivin.1 inactivator-kallikrein (C.hivin.1In-K)  
complexes. The formation of complexes assayed by this method  
paralleled the inhibition of [human] plasma  
kallikrein esterase activity by C.hivin.1 inactivator in purified  
systems. C.hivin.1In-K complexes were detected when a final concentration  
of 5.7 nM plasma kallikrein was added to plasma, equivalent to the  
activation of 1% of the plasma prekallikrein. Exogenous Hageman factor  
fragment added to plasma induced the rapid formation of C.hivin.1In-K  
complexes, whereas there was an appreciable delay when the plasma contact  
system was activated by the addition of kaolin. In both systems, the rate  
of formation and final amount of complex generated were directly related  
to the concentration of Hageman factor fragment or of kaolin added,  
indicating that this proteolytic pathway is tightly regulated.  
C.hivin.1In-K complexes were not generated by kaolin in plasma  
congenitally deficient in Hageman factor or prekallikrein or by kallikrein  
in hereditary angioedema plasma deficient in C.hivin.1 inactivator, thus  
confirming the specificity of the assay. Sucrose gradient  
ultracentrifugation studies showed plasma C.hivin.1In-K complexes to have  
a MW consistent with a 1:1 molar complex. In contrast, the complex  
displayed an anomalously high MW on gel filtration chromatography. A  
sensitive and specific probe was developed for documenting plasma  
kallikrein activation.

L5 ANSWER 16 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN

AN 1983:402149 CAPLUS

DN 99:2149

OREF 99:443a,446a

TI Biochemistry of human tissue kallikrein

AU Geiger, R.; Hofmann, W.; Franke, M.; Baur, X.

CS Chir. Klin. Innenstadt, Univ. Munchen, Munich, Fed. Rep. Ger.

SO Advances in Experimental Medicine and Biology (1983), 156A(Kinins-3, Pt.  
A), 275-88

CODEN: AEMBAP; ISSN: 0065-2598

DT Journal

LA English

AB Kallikrein (I) from human urine, pancreas, large intestine, blood plasma,  
and seminal plasma were studied. Procedures for the isolation of I from  
these sources are reviewed; amino acid compns. and mol. wts. were determined  
The various I forms have similar properties with respect to cleavage of  
synthetic substrates, kinin-liberating and blood pressure-lowering  
potency, aprotinin complex formation, and inhibition by  
diisopropylfluorophosphate. A sensitive and specific immunoassay for  
urinary I was developed which used human anti-I IgG and peroxidase; the  
procedure could be used for determination of other human tissue I levels.

Urinary

I was inhibited progressively by increasing concns. of purified human  
 $\alpha$ 1-antitrypsin and serum. Serum from a patient with Z/Z  
 $\alpha$ 1-antitrypsin deficiency did not inhibit I; apparently,  
 $\alpha$ 1antitrypsin is the only glandular I inhibitor in serum.

L5 ANSWER 17 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on  
STN  
AN 1984:97758 BIOSIS  
DN PREV198427014250; BR27:14250  
TI INHIBITION OF HUMAN PLASMA KALLIKREIN BY  
COMPLEMENT C-1 ESTERASE INHIBITOR DETERMINATION OF  
KINETIC MECHANISM AND RATE CONSTANTS.  
AU NILSSON T [Reprint author]  
CS DEP CLINICAL CHEMISTRY, UMEA UNIV HOSP, S-901 85 UMEA, SWEDEN  
SO Thrombosis and Haemostasis, (1983) Vol. 50, No. 1, pp. 229.  
Meeting Info.: 9TH INTERNATIONAL CONGRESS ON THROMBOSIS AND HEMOSTASIS,  
JULY 4-8, 1983. THROMB HEMOSTASIS.  
CODEN: THHADQ. ISSN: 0340-6245.  
DT Conference; (Meeting)  
FS BR  
LA ENGLISH

L5 ANSWER 18 OF 22 MEDLINE on STN DUPLICATE 3  
AN 82153809 MEDLINE  
DN PubMed ID: 6917564  
TI Simple chromogenic peptide substrate assays for determining  
prekallikrein, kallikrein inhibition and kallikrein  
"like" activity in human plasma.  
AU Gallimore M J; Friberger P  
SO Thrombosis research, (1982 Feb 1) Vol. 25, No. 3, pp. 293-8.  
Journal code: 0326377. ISSN: 0049-3848.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 198205  
ED Entered STN: 17 Mar 1990  
Last Updated on STN: 3 Mar 2000  
Entered Medline: 21 May 1982

L5 ANSWER 19 OF 22 EMBASE COPYRIGHT (c) 2008 Elsevier B.V. All rights  
reserved on STN  
AN 1981233530 EMBASE  
TI Inhibition of human tissue (urinary)  
kallikrein by sera of patients suffering from hereditary  
 $\alpha(1)$ -antitrypsin ( $\alpha(1)$ -proteinase inhibitor) deficiency.  
AU Geiger, R.; Koenig, G.; Fruhmans, G.  
CS Abt. Klin. Chem., Univ. Munchen, Germany.  
SO Hoppe-Seyler's Zeitschrift fur Physiologische Chemie, (1981) Vol. 362, No.  
7, pp. 1013-1015.  
ISSN: 0018-4888 CODEN: HSZPAZ  
CY Germany  
DT Journal; Article  
FS 029 Clinical and Experimental Biochemistry  
LA English  
SL German  
ED Entered STN: 9 Dec 1991  
Last Updated on STN: 9 Dec 1991  
AB Sera of patients who present a hereditary  $\alpha(1)$  antitrypsin deficiency  
were tested as to their capacity to inhibit  
human tissue (urinary) kallikrein. The degree of the  
progressive inhibition was dependent on the nature of the genetically  
conditioned  $\alpha(1)$  antitrypsin deficiency. Sera of homozygote  $\alpha(1)$   
type Z/Z showed no inhibitory action; sera of heterozygote type S/Z a  
diminished inhibitory action in comparison with normal sera.

L5 ANSWER 20 OF 22 BIOSIS COPYRIGHT (c) 2008 The Thomson Corporation on  
STN  
AN 1982:170697 BIOSIS  
DN PREV198273030681; BA73:30681  
TI CLINICAL IMPORTANCE OF EXAMINING BLOOD KININS AND INSULIN IN PANCREATITIS.  
AU ORLOV V A [Reprint author]; VEDROVA N N  
CS DIV HOSP THER SUBORD, BASE CLIN HOSP NO 20, MOSCOW, USSR  
SO Terapevticheskii Arkhiv, (1981) Vol. 53, No. 2, pp. 45-48.  
CODEN: TEARAI. ISSN: 0040-3660.  
DT Article  
FS BA  
LA RUSSIAN  
AB The content of prekallikrein, the kallikrein inhibitor and kallikrein, as well as the spontaneous esterase activity, the content of kininogen, the blood kininase activity and the pancreatic secretory function were studied in 104 patients with various forms of pancreatitis. The studies were based on the results of the i.v. glucose tolerance test and examination of the immunoreactive insulin content in the blood. During the acute stage of pancreatitis, the kininogenesis was intensified because of disturbed equilibrium in the kallikrein/kallikrein inhibitor system, while the level of kininogen (the kinin precursor) was lowered. Disturbances of the pancreatic secretory function were revealed almost in 90% of the patients with pancreatitis. The program of examining patients with pancreatitis should thus include examinations of the blood kinin system and insulin.

L5 ANSWER 21 OF 22 MEDLINE on STN DPLICATE 4  
AN 77140856 MEDLINE  
DN PubMed ID: 14935  
TI Kallikrein inhibitors in rat plasma.  
AU Hojima Y; Isobe M; Moriya H  
SO Journal of biochemistry, (1977 Jan) Vol. 81, No. 1, pp. 37-46.  
Journal code: 0376600. ISSN: 0021-924X.  
CY Japan  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 197705  
ED Entered STN: 13 Mar 1990  
Last Updated on STN: 3 Mar 2000  
Entered Medline: 25 May 1977  
AB The kallikrein inhibitor contents of human and animal plasma were determined with glandular kallikreins [EC 3.4.21.8]. One ml of plasma could inactivate 20-700 kallikrein units (KU). Rat plasma was the most potent and inactivated 230-700 KU. However, no enzyme capable of inactivating kallikrein could be found in this plasma. Two fractions which inhibited hog pancreatic kallikrein, a fraction corresponding to alpha2-macroglobulin and a fraction which was eluted prior to albumin, were separated from rat plasma by Sephadex G-200 gel filtration. The former inhibitor could inhibit hog pancreatic kallikrein action on Nalpha-benzoyl-L-arginine ethyl ester (BAEE) as well as in the dog vasodilator assay. The other inhibitor was partially purified from rat plasma. One mg of the preparation inhibited 67 KU and the hydrolysis of 5.8 micromoles/min of BAEE by hog pancreatic kallikrein [EC 3.4.21.8]. The inhibitor also inhibited other glandular and plasma kallikreins, trypsin [EC 3.4.21.4], alpha-chymotrypsin [EC 3.4.21.1], etc. The optimal pH of the inhibitor was 7.5-8. The inhibitor was unstable below pH 5, and was destroyed by heating at temperature above 60 degrees. The isoelectric point of the inhibitor was determined by Ampholine focusing to be 4.4, and its molecular weight was estimated to be 73,000 by Sephadex G-100 and G-150 filtrations. Several experimental results

suggested that this inhibitor differed from alphas<sub>1</sub>-antitrypsin.

L5 ANSWER 22 OF 22 CAPLUS COPYRIGHT 2008 ACS on STN

AN 1978:4252 CAPLUS

DN 88:4252

OREF 88:763a,766a

TI Determination of "contact" prekallikrein and kallikrein inhibitor in human blood plasma

AU Veremeenko, K. N.; Volokhonskaya, L. I.; Kizim, A. I.; Lositskaya, V. M.; Meged, N. F.; Gontar, A. M.

CS Kiev. Nauchno-Issled. Inst. Stolaringol., Kiev, USSR

SO Kininy Kininovaya Sist. Krovi (1976), 62-3. Editor(s): Paskhina, T. S.; Men'shikov, V. V. Publisher: Pervyi Mosk. Med. Inst., Moscow, USSR.

CODEN: 36SHAQ

DT Conference

LA Russian

AB Plasma prekallikrein was activated by contact with SiO<sub>2</sub> or kaolin and the resulting proteolytic activity (determined by the release of arginine from protamine sulfate) could be inhibited by Trasylol or its analogs, soybean trypsin inhibitor, and  $\alpha$ 2-macroglobulin but was not affected by ovomucoid. Thus, the activity is due to kallikrein and not to plasmin. In the blood of patients with acute hepatitis the levels of kallikrein inhibitors and prekallikrein decreased below normal. The proteins, apparently formed in liver, can be used as sensitive indicators of hepatic function. In acute pancreatitis the levels of prekallikrein decreased and those of kallikrein and antitrypsin as well as antitrypsin-to- $\alpha$ 2-macroglobulin ratio increased above normal. After treatment the ratio decreased. The contact determination of prekallikrein and kallikrein inhibitors

is simple and reproducible and therefore suitable for clin. use.

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